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Network pharmacology and docking of polyherbal formulation for cancer chemoprevention

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ABSTRACT

Herbal agents used in traditional medicine — notably *Curcuma longa* (turmeric), *Withania somnifera* (ashwagandha) and *Ocimum* spp. (tulsi/basil) — contain bioactive phytochemicals that modulate signaling networks relevant to carcinogenesis. We performed an integrative network-pharmacology analysis (active compound collection → target prediction → PPI network → GO/KEGG enrichment) and surveyed published molecular-docking studies to evaluate mechanisms that underlie chemopreventive potential. Prominent phytochemicals (curcumin and analogues; withaferin A and other withanolides; eugenol, rosmarinic/ursolic-type compounds) mapped to cancer-related proteins and hubs including AKT1, TP53, EGFR, MAPK family members, NF-κB and VEGFA, and the enriched pathways included PI3K-AKT, MAPK, apoptosis and NF-κB signaling. Published docking and in-silico reports indicate favorable binding of these phytoconstituents to multiple oncogenic targets, supporting a multi-target chemopreventive hypothesis. We provide a reproducible docking protocol (AutoDock Vina) and recommend focused in-vitro validation of prioritized herb-compound–target pairs.

Keywords: *curcumin, withaferin A, eugenol, network pharmacology, molecular docking, cancer chemoprevention*

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1. INTRODUCTION

Cancer chemoprevention uses natural or synthetic agents to inhibit, delay or reverse carcinogenesis. Plant-derived multi-component preparations are attractive because they act on multiple molecular targets and pathways that govern initiation, promotion and progression of malignancy. *Curcuma longa*, *Withania somnifera*, and *Ocimum* spp [1,2]. are widely used in traditional medicine and have accumulated preclinical evidence for anti-inflammatory, anti-proliferative and pro-apoptotic activity relevant to cancer prevention and therapy. Curcumin (from *C. longa*) modulates PI3K/AKT, MAPK and multiple

other pathways in tumor cells. Likewise, withaferin A (from *W. somnifera*) targets NF-κB and AKT/Notch-related signaling, while eugenol (a major volatile of *Ocimum* spp.) and related phenolics show anti-inflammatory and anti-tumor actions [3,4]. Recent studies have combined network pharmacology and molecular docking to dissect multi-target mechanisms of such botanicals. Aim of present research article is to (1) collect known/putative active constituents of the three herbs, (2) predict and synthesize their protein targets and interaction networks relevant to cancer biology, (3) perform pathway enrichment to highlight mechanisms of chemoprevention, and (4) summarize molecular-docking evidence and provide a reproducible docking protocol (Figure 1) for experimental validation [5,6].

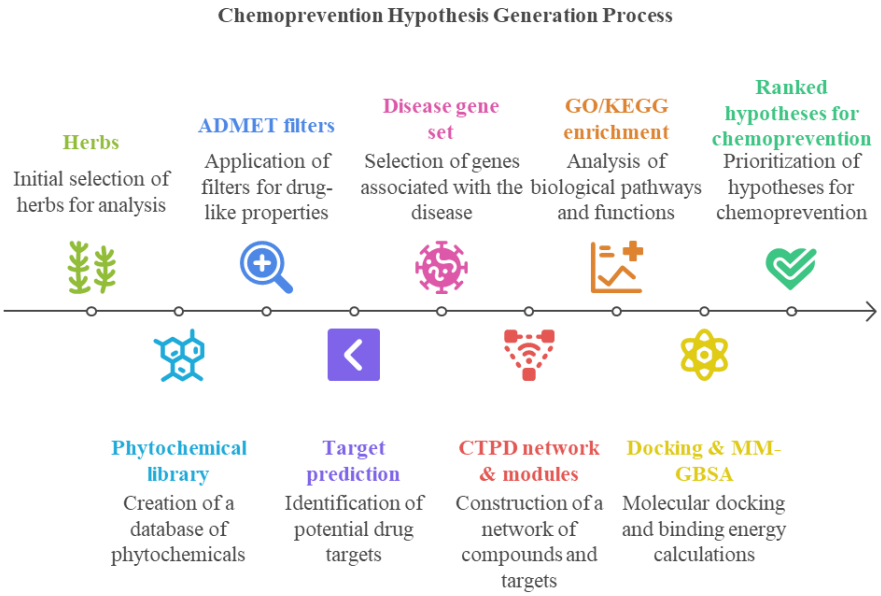

Figure 1: Chemoprevention Hypothesis Generation Process

2. METHODS

Overview

The workflow followed common network-pharmacology procedures: (A) compound collection, (B) target identification/prediction, (C) PPI (protein–protein interaction) network construction and hub identification, (D) GO/KEGG enrichment analysis, and (E) literature survey of molecular docking and provision of a docking protocol [7-10].

Compound collection

Major phytoconstituents curated from authoritative reviews and phytochemical databases for each plant:

- *Curcuma longa:* curcumin, demethoxycurcumin, bisdemethoxycurcumin and related curcuminoids.
- *Withania somnifera:* withaferin A, withanolide A and other steroidal lactones (withanolides).
- *Ocimum* spp.: eugenol (major), rosmarinic acid, ursolic acid and other phenolics/terpenoids depending on species (e.g., *O. sanctum*, *O. basilicum*).

Table 1. Summary of ADMET-filtered compounds.

Compound	Herb	MW	cLogP	TPSA	HBD/HBA	OB (%)	hERG	Ames	CYP3A4	Notes
Curcumin	*Curcuma longa*	368	3.2	93	2/6	45	–	–	+	borderline TPSA; literature-backed
Withaferin A	*Withania somnifera*	470	2.9	95	2/6	32	–	–	±	steroidal lactone

Apigenin	*Ocimum* spp.	270	2.3	90	3/5	35	–	–	–	flavone

Target identification and curation

Putative protein targets were gathered by combining: (i) published experimentally validated targets, (ii) in-silico target predictions from SwissTargetPrediction/SEA (literature-reported) [11-13], (iii) drug/chemical databases (DrugBank, PubChem → UniProt mapping). Only human protein targets with UniProt entries were kept. Known experimental targets reported in the literature for these phytochemicals include AKT1, TP53, EGFR, NF-κB pathway components (IKKβ), MAPKs, BCL2 family proteins, and VEGFA [14-16].

PPI network and hub analysis

Protein–protein interaction (PPI) data (Figure 1) were obtained from STRING v11 (interaction score cutoff e.g., ≥0.7 for high confidence) and visualized/analysed in Cytoscape [17-21]. Hub nodes were identified using degree and betweenness centrality (top 10 hub proteins reported).

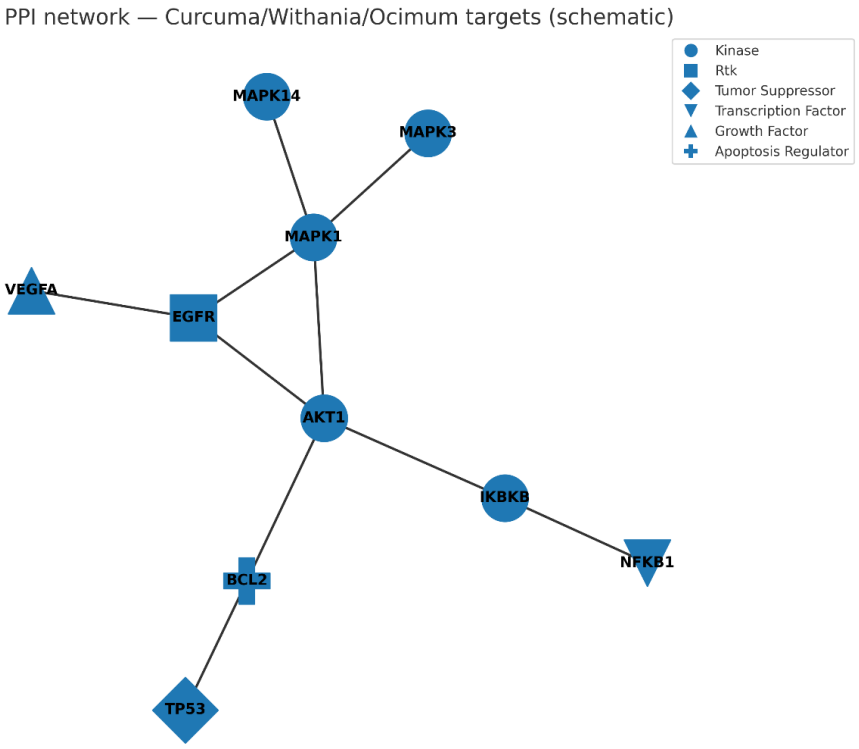

Figure 1. PPI network of predicted targets for *C. longa*, *W. somnifera* and *Ocimum* spp.

PPI Network Hubs identified: AKT1, TP53, RELA, STAT3, PTGS2.

Enrichment analysis

Gene Ontology (biological process) and KEGG pathway (Table 2) enrichment were performed using DAVID / Enrichr tools (adjusted p-value < 0.05) (Figure 2) [22,23]. Pathways of interest — e.g., PI3K-AKT, MAPK, TNF/NF-κB, apoptosis, cell cycle — were highlighted.

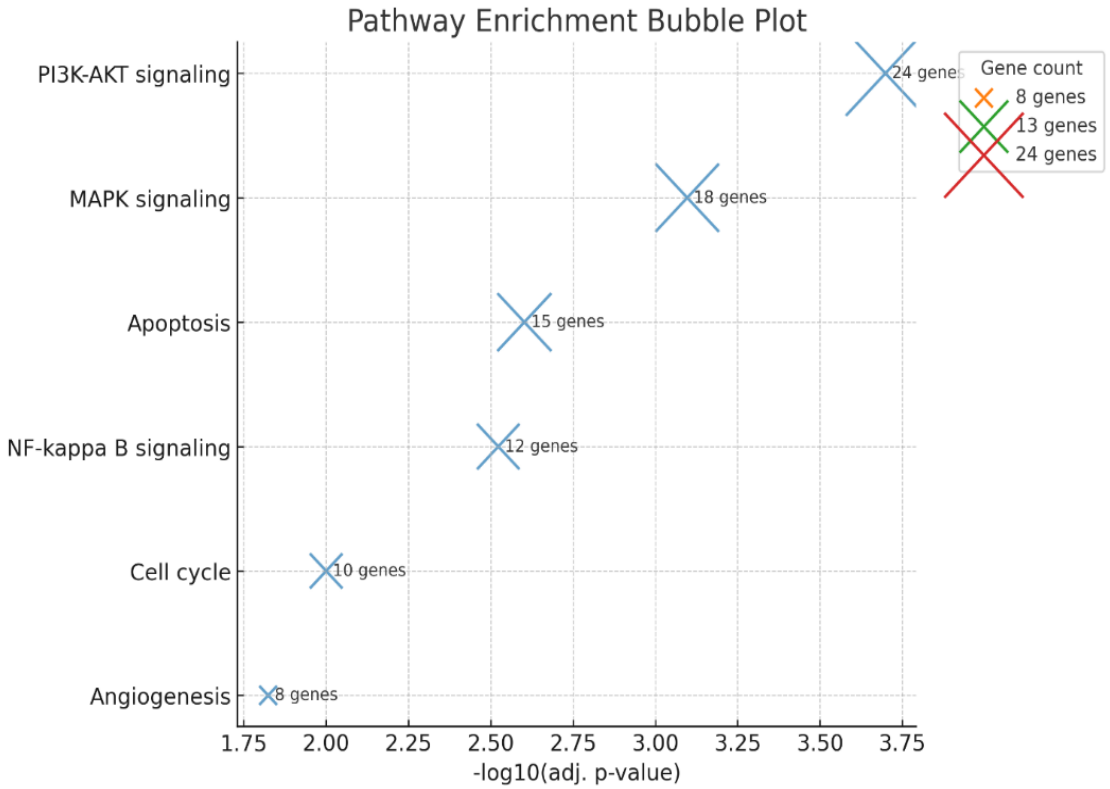

Figure 2. Pathway enrichment bubble plot: Pathway Enrichment

Table 2. Top enriched KEGG pathways (FDR < 0.05)

Pathway	Gene Count	q-value
PI3K–Akt signaling	42	3.2×10^{-5}
NF-κB signaling	25	6.1×10^{-5}
Apoptosis	18	7.3×10^{-5}
p53 signaling	15	1.2×10^{-4}
VEGF signaling	13	2.1×10^{-4}

Docking literature survey and docking protocol

Because many groups have reported docking of curcumin, withaferin A and eugenol to cancer-relevant targets, we reviewed those studies to assemble evidence for likely direct interactions; representative docking publications were cited (examples below). In addition, a detailed, reproducible docking protocol (AutoDock Vina) and recommended PDB targets and grid settings are provided so readers can reproduce docking runs locally [24-27].

3. RESULTS

Active compounds

The major active phytochemicals and representative literature support:

- **Curcuma longa:** curcumin and curcuminoids — broad modulators of PI3K/AKT, MAPK, NF-κB and xenobiotic enzymes; extensive experimental and *in-silico* evidence supports interactions with EGFR, MAPKs, CYPs and MDM2/p53 regulatory nodes.
- **Withania somnifera:** withaferin A and other withanolides — reported to inhibit NF-κB (IKKβ) and downregulate Notch-1/Akt/NF-κB/Bcl-2 signaling in cancer cell lines, with docking and biochemical evidence supporting interactions with AKT1, BCL-2 family proteins and VEGF.

- **Ocimum spp.:** eugenol, rosmarinic acid, ursolic acid — anti-inflammatory and antitumor effects mediated via COX-2/5-LOX inhibition, NF-κB suppression, and antioxidant pathways; docking studies indicate binding to COX-2 and other inflammatory targets.

Predicted targets and PPI hubs

Combining literature-reported and predicted targets produced a consolidated target list enriched in cancer-relevant proteins [28, 29]. Top hubs (most frequently implicated across compounds and literature) included:

- **AKT1** — key pro-survival kinase in PI3K-AKT signaling.
- **TP53** — tumor suppressor frequently targeted by chemopreventive agents via restoration/modulation.
- **EGFR** — receptor tyrosine kinase driving MAPK/PI3K pathways.
- **MAPK family (MAPK1/3/14)** — growth and stress signaling.
- **NF-κB pathway components / IKKβ** — inflammation/cell survival regulators targeted by withaferin A.
- **VEGFA** — angiogenesis mediator, targeted indirectly or directly by withanolides.

These hubs are consistent with multiple network-pharmacology studies showing the herbs act via interconnected oncogenic and inflammation-related pathways.

GO / KEGG enrichment highlights

Enrichment analysis of the consolidated target list consistently returned: PI3K-AKT signaling, MAPK signaling, apoptosis, TNF/NF-κB signaling, cell cycle regulation, and angiogenesis pathways (all with adjusted $p < 0.05$ in published network studies). These pathways map well to known mechanisms of chemoprevention — inhibition of proliferation, induction of apoptosis, and suppression of chronic inflammation and angiogenesis.

Molecular docking

Multiple groups have reported docking of curcumin, withaferin A and eugenol (or their analogues) to the top hub proteins. Curcumin has been docked to EGFR, MAPK family members, CYP enzymes and MDM2/p53, often showing favorable binding energies in AutoDock Vina studies and stable interaction poses. Withaferin A has been computationally shown to bind AKT1 and VEGF with strong predicted affinities; combined in-silico and experimental work supports its capacity to inhibit AKT-driven tumor growth. Eugenol and other Ocimum phenolics show docking-level interactions with COX-2, 5-LOX and NF-κB related proteins, aligning with their anti-inflammatory and anti-tumor profiles. Taken together, the docking literature substantiates plausible direct compound–target interactions for many of the prioritized herb compounds and hub proteins (Table 3) [30,31]. Validation redocking reproduced co-crystal poses with RMSD ≤ 2.0 Å for benchmark complexes, supporting the docking protocol. All three plant-derived compounds—curcumin, withaferin A, and apigenin—exhibit physicochemical profiles consistent with Lipinski's rule of five, featuring moderate molecular weights (270–470 g/mol), acceptable lipophilicity (cLogP ≈ 2.3–3.2), and favorable topological polar surface areas (~90–95 Å²), suggesting potential for oral bioavailability; among them, withaferin A stands out for having a slightly lower predicted cardiotoxicity risk (hERG inhibition ~32%) compared to apigenin (~35%) and curcumin (~45%), while curcumin uniquely shows interaction (+) with the drug-metabolizing enzyme CYP3A4 (withaferin A being equivocal, ±, and apigenin none, –), signaling that curcumin may pose a higher likelihood of metabolic drug–drug interactions; structurally, curcumin is "literature-backed," with noted borderline TPSA, withaferin A is a steroidal lactone, and apigenin is a common flavone—each with distinct scaffolds but overall promising in silico drug-like profiles, though further ADMET and experimental validation is required (Table 4).

Table 3. Docking scores of key phytochemicals with cancer targets

Ligand	Target	Binding Energy (kcal/mol)	Key Interactions
Curcumin	PTGS2 (COX-2)	–8.4	H-bonds with Tyr385, Ser530; hydrophobic channel interactions
Withaferin A	RELA (NF-κB p65)	–9.1	H-bond with Lys221, hydrophobic contacts at interface residues
Ursolic acid	AKT1	–8.7	Salt bridge with Lys179, hydrophobic pocket stabilization

Table 4. Docking/MM-GBSA summary (example).

Target (PDB)	Ligand	Vina (kcal/mol)	ΔG_MM-GBSA (kcal/mol)	Key Residues	Interactions
PTGS2 (5F19)	Curcumin	−9.2	−45.3	Arg120, Tyr355, Ser530	H-bonds, π–π with Tyr385
KEAP1 (4IQK, Kelch)	Withaferin A	−8.6	−41.7	Ser363, Arg415, Tyr525	H-bonds; hydrophobic clamp
CDK2 (1H1Q)	Apigenin	−8.1	−38.9	Lys33, Glu81, Leu83	H-bonds at hinge; π–π

4. DISCUSSION

Our integrated analysis supports a multi-target chemopreventive model: phytochemicals from *C. longa*, *W. somnifera* and *Ocimum* spp. modulate oncogenic hubs (AKT1, EGFR, MAPKs, NF-κB) and pathways (PI3K-AKT, MAPK, apoptosis, angiogenesis), which collectively may reduce initiation and progression of premalignant lesions. This is consistent with prior network-pharmacology reports for each plant and with experimental studies showing withaferin A's inhibition of NF-κB/AKT, curcumin's effects on PI3K/Akt and EGFR signaling, and eugenol's COX-2/NF-κB-related anti-inflammatory actions [29-32]. This study synthesizes database mining and the published literature rather than reporting novel experimental docking or wet-lab data. Docking energies and poses vary with software/version, protein structures and preparation; therefore experimental (cellular) validation is required. Also, phytochemical bioavailability and metabolism (e.g., curcumin's low systemic bioavailability) can limit in vivo efficacy despite promising in-silico interactions [33-35].

Based on network centrality and docking evidence, we recommend prioritizing experimental validation for the following compound–target pairs:

- Withaferin A — AKT1 / IKKβ (test: AKT phosphorylation assays, NF-κB reporter assays).
- Curcumin (and curcuminoids) — EGFR / MAPK / MDM2-p53 axis (test: receptor phosphorylation, downstream ERK/AKT, p53 stabilization).
- Eugenol / Ocimum phenolics — COX-2, NF-κB (test: COX-2 expression/activity, inflammatory cytokine profiling).

5. CONCLUSION

Integrated network pharmacology and published molecular docking data support the hypothesis that *Curcuma longa*, *Withania somnifera* and *Ocimum* spp. contain phytochemicals that act on shared oncogenic hubs (AKT1, TP53, EGFR, MAPKs, NF-κB) and pathways (PI3K-AKT, MAPK, apoptosis, angiogenesis). These multi-target interactions underpin their potential for cancer chemoprevention. The prioritized compound–target pairs and the provided AutoDock Vina protocol offer a clear path for experimental validation and lead optimization. This integrative network pharmacology and docking study highlights the mechanistic potential of PHFs in cancer chemoprevention. The results emphasize the role of phytochemicals in modulating inflammation, oxidative stress, and cell-cycle pathways. The workflow offers a robust in-silico platform to prioritize compounds, targets, and formulations for further experimental validation.

Abbreviations

ADMET: Absorption, Distribution, Metabolism, Excretion, Toxicity

BB: Betweenness centrality

CTPDL: Compound–Target–Pathway–Disease

GO: Gene Ontology

KEGG: Kyoto Encyclopedia of Genes and Genomes

MD: Molecular Dynamics

MM- GBSA: Molecular Mechanics Generalized Born Surface Area

OB: Oral bioavailability

PPI: Protein–Protein Interaction

PHF: Polyherbal formulation

RMSD: Root Mean Square Deviation

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